PATENT
Customer No. 22,852
Attorney Docket No. 04853.0085

IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

In re Application of:	
OGATA et al.	Group Art Unit: 1644
Serial No.: 10/019,501	Examiner: Phuong Huynh
§ 371 Date: December 31, 2001	
PCT Filing Date: July 3, 2000	
For: AGENT FOR AMELIORATING LOW VASOPRESSIN LEVEL	

Commissioner for Patents
P.O. Box 1450
Alexandria, VA 22313-1450

Sir:

RESPONSE TO THE RESTRICTION REQUIREMENT

In a restriction requirement dated December 3, 2003, the Examiner required restriction under PCT Rule 13.1 between the following groups:

Group I: claims 1-11, drawn to a method of maintaining or increasing low vasopressin level comprising administering to a patient at least one substance that inhibits the binding between PTHrP and a receptor thereof.

Group II: claims 12-13, drawn to a method of treating hyperosmolarity.

Group III: claims 14-15, drawn to a method of treating dehydration.

Group IV: claims 16-22, drawn to a method of inhibiting the binding between PTHrP and a receptor thereof.

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Applicants provisionally elect to prosecute Group I, claims 1-11 drawn to a method of maintaining or increasing low vasopressin level comprising administering to a patient at least one substance that inhibits the binding between PTHrP and a receptor thereof, with traverse.

In making the restriction requirement, the Examiner stated that the inventions are unrelated as the prior art, *Yamamoto et al.*, Endocrinology 138(5):2066-2072 (1997), allegedly teaches a method of lowering low vasopressin level using a truncated form of PTHrP as a PTHrP receptor antagonist.

Applicants disagree. The common technical feature presented in the claims of this application is the fact that the claims cover methods of maintaining or increasing low vasopressin level via inhibition of the binding between PTHrP and a receptor thereof. Further, diseases and disorders caused by low vasopressin level may be treated in this fashion, by maintaining or increasing the vasopressin level. Yamamoto et al. use an *in vitro* system to characterize the effects of a fragment of PTHrP on the rat supraoptic nucleus (SON). The coronal hypothalamic slices containing the SON are artificially perfused with the PTHrP fragment. Further, a receptor for PTHrP that mediates this effect, which the authors allege is "distinct" from already identified PTHrP receptors, has not been identified. Thus, any physiological importance of this effect is unclear and, in fact, may not exist.

PTHrP and its interaction between type I and type II PTH/PTHrP receptors, on the other hand, is well known. Various physiological roles for PTHrP via these receptors have also been characterized. PTHrP "has been identified in tumors

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associated with humoral hypercalcemia of malignancy." Yamamoto et al. at 2066, left column, first sentence. Further, Applicants have demonstrated that levels of vasopressin, which are below normal in a hypercalcemia model, can be increased using antibodies against PTHrP. See Specification, Examples 1 and 2, pages 19-23. Applicants respectfully submit that the claims share the same proven special technical feature and therefore request that the restriction requirement be withdrawn.

Please grant any extensions of time required to enter this response and charge any additional required fees to our deposit account 06-0916.

Respectfully submitted,

FINNEGAN, HENDERSON, FARABOW, GARRETT & DUNNER, L.L.P.

Dated: February 3, 2004

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